

WEST Search History

DATE: Monday, November 14, 2005

Hide?	Set Name	Query	Hit Count
		<i>DB=PGPB,USPT,USOC,EPAB,JPAB,DWPI,TDBD; THES=ASSIGNEE; PLUR=YES; OP=ADJ</i>	
<input type="checkbox"/>	L30	L28 and prostate	2
<input type="checkbox"/>	L29	L28 and melanoma	2
<input type="checkbox"/>	L28	20020039754	2
<input type="checkbox"/>	L27	Fruehauf-john.in.	5
<input type="checkbox"/>	L26	taylor-clive-r.in.	14
<input type="checkbox"/>	L25	skinner-donald-g.in.	0
<input type="checkbox"/>	L24	groshen-susan.in.	0
<input type="checkbox"/>	L23	esrig-david.in.	0
<input type="checkbox"/>	L22	bochner-bernard-h.in.	0
<input type="checkbox"/>	L21	stein-john-p.in.	7
<input type="checkbox"/>	L20	ginsberg-david-a.in.	0
<input type="checkbox"/>	L19	grossfeld-gary-d.in.	0
<input type="checkbox"/>	L18	cote-richard-j.in.	6
<input type="checkbox"/>	L17	cote-r.in.	30
<input type="checkbox"/>	L16	Bouck-noel-p.in.	15
<input type="checkbox"/>	L15	L14 and l10	316
<input type="checkbox"/>	L14	L13 and l11	330
<input type="checkbox"/>	L13	p53	157705
<input type="checkbox"/>	L12	Lp53	10
<input type="checkbox"/>	L11	TSP-1	515
<input type="checkbox"/>	L10	angiogenesis	26625
<input type="checkbox"/>	L9	L8 and angiogenesis	318
<input type="checkbox"/>	L8	L7 and l3	342
<input type="checkbox"/>	L7	thrombospondin-1	659
<input type="checkbox"/>	L6	bouck-n.in.	0
<input type="checkbox"/>	L5	Dameron-k.in.	0
<input type="checkbox"/>	L4	L2 and l3	57
<input type="checkbox"/>	L3	p53	157705
<input type="checkbox"/>	L2	brawer	208
<input type="checkbox"/>	L1	brawer-mk.in.	0

END OF SEARCH HISTORY

Refine Search

Search Results -

Terms	Documents
10734880	0

Database:

US Pre-Grant Publication Full-Text Database
 US Patents Full-Text Database
 US OCR Full-Text Database
 EPO Abstracts Database
 JPO Abstracts Database
 Derwent World Patents Index
 IBM Technical Disclosure Bulletins

Search:

L6

Search History

DATE: Monday, November 14, 2005 [Printable Copy](#) [Create Case](#)

<u>Set Name</u> side by side	<u>Query</u>	<u>Hit Count</u>	<u>Set Name</u> result set
<i>DB=PGPB,USPT,USOC,EPAB,JPAB,DWPI,TDBD; THES=ASSIGNEE; PLUR=YES; OP=ADJ</i>			
<u>L6</u>	10734880	0	<u>L6</u>
<u>L5</u>	10295188	3	<u>L5</u>
<u>L4</u>	10144142	4	<u>L4</u>
<u>L3</u>	fruehauf-john.in.	5	<u>L3</u>
<u>L2</u>	5840507.pn.	2	<u>L2</u>
<u>L1</u>	6303324.pn.	2	<u>L1</u>

END OF SEARCH HISTORY

Freeform Search

Database:	US Pre-Grant Publication Full-Text Database
	US Patents Full-Text Database
	US OCR Full-Text Database
	EPO Abstracts Database
	JPO Abstracts Database
	Derwent World Patents Index
	IBM Technical Disclosure Bulletins

Term:	L8 and angiogenesis	▲
		▼

Display:	10	Documents in Display Format:	-	Starting with Number	1
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Generate: ☐ Hit List ☒ Hit Count ☐ Side by Side ☐ Image

Search

Clear

Interrupt

Search History

DATE: Monday, November 14, 2005 [Printable Copy](#) [Create Case](#)

<u>Set Name</u> side by side	<u>Query</u>	<u>Hit Count</u>	<u>Set Name</u> result set
<i>DB=PGPB,USPT,USOC,EPAB,JPAB,DWPI,TDBD; THES=ASSIGNEE; PLUR=YES; OP=ADJ</i>			
<u>L9</u>	L8 and angiogenesis	318	<u>L9</u>
<u>L8</u>	L7 and l3	342	<u>L8</u>
<u>L7</u>	thrombospondin-1	659	<u>L7</u>
<u>L6</u>	bouck-n.in.	0	<u>L6</u>
<u>L5</u>	Dameron-k.in.	0	<u>L5</u>
<u>L4</u>	L2 and l3	57	<u>L4</u>
<u>L3</u>	p53	157705	<u>L3</u>
<u>L2</u>	brawer	208	<u>L2</u>
<u>L1</u>	brawer-mk.in.	0	<u>L1</u>

END OF SEARCH HISTORY

Connecting via Winsock to STN

Welcome to STN International! Enter x:x

LOGINID:ssptadhh1642

PASSWORD:

TERMINAL (ENTER 1, 2, 3, OR ?):2

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NEWS 12 OCT 17 STN(R) AnaVist(TM), Version 1.01, allows the export/download
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NEWS 15 OCT 27 EPFULL enhanced with additional content

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FILE 'HOME' ENTERED AT 14:05:42 ON 14 NOV 2005

=> FIL MEDLINE, BIOSIS, EMBASE

COST IN U.S. DOLLARS

SINCE FILE
ENTRY

TOTAL
SESSION

FULL ESTIMATED COST

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0.21

FILE 'MEDLINE' ENTERED AT 14:05:55 ON 14 NOV 2005

FILE 'BIOSIS' ENTERED AT 14:05:55 ON 14 NOV 2005

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FILE 'EMBASE' ENTERED AT 14:05:55 ON 14 NOV 2005

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=> s p53

L1 114062 P53

=> s thrombospondin-1

L2 3207 THROMBOSPONDIN-1

=> s angiogenesis

L3 81008 ANGIOGENESIS

=> s l1 and l2

L4 244 L1 AND L2

=> s l3 and l4

L5 183 L3 AND L4

=> duplicate remove

ENTER L# LIST OR (END):15

DUPLICATE PREFERENCE IS 'MEDLINE, BIOSIS, EMBASE'

KEEP DUPLICATES FROM MORE THAN ONE FILE? Y/(N):n

PROCESSING COMPLETED FOR L5

L6 106 DUPLICATE REMOVE L5 (77 DUPLICATES REMOVED)

=> s breast cancer

L7 291994 BREAST CANCER

=> l6 and l7

L6 IS NOT A RECOGNIZED COMMAND

The previous command name entered was not recognized by the system.

For a list of commands available to you in the current file, enter

"HELP COMMANDS" at an arrow prompt (=>).

=> s l6 and l7

L8 14 L6 AND L7

=> s prostate cancer

L9 103114 PROSTATE CANCER

=> s l6 and l9

L10 6 L6 AND L9

=> s melanoma

L11 170286 MELANOMA

=> s l6 and l11

L12 9 L6 AND L11

=> s l8 or l10

L13 18 L8 OR L10

=> s l13 or l12

L14 26 L13 OR L12

=> display l14

ENTER ANSWER NUMBER OR RANGE (1):1-26

ENTER DISPLAY FORMAT (FILEDEFAULT):all

L14 ANSWER 1 OF 26 MEDLINE on STN
 AN 2002182450 MEDLINE
 DN PubMed ID: 11916242
 TI Aerosol delivery of PEI-p53 complexes inhibits B16-F10 lung metastases through regulation of angiogenesis.
 AU Gautam Ajay; Densmore Charles L; Melton Sara; Golunski Eva; Waldrep J Clifford
 CS Department of Molecular Physiology and Biophysics, Baylor College of Medicine, Houston, Texas 77030, USA.
 SO Cancer gene therapy, (2002 Jan) 9 (1) 28-36.
 Journal code: 9432230. ISSN: 0929-1903.
 CY England: United Kingdom
 DT Journal; Article; (JOURNAL ARTICLE)
 LA English
 FS Priority Journals
 EM 200207
 ED Entered STN: 20020403
 Last Updated on STN: 20020710
 Entered Medline: 20020709
 AB Inhibition of pulmonary metastases poses a difficult clinical challenge for current therapeutic regimens. We have developed an aerosol system utilizing a cationic polymer, polyethyleneimine (PEI), for topical gene delivery to the lungs as a novel approach for treatment of lung cancer. Using a B16-F10 murine melanoma model in C57BL/6 mice, we previously demonstrated that aerosol delivery of PEI-p53 DNA resulted in highly significant reductions in the tumor burden ($P < .001$) in treated animals, and also lead to about 50% increase in the mean length of survival of the mice-bearing B16-F10 lung tumors. The mechanisms of this antitumor effect of p53 are investigated in this report. Here, we demonstrate that the p53 transfection leads to an up-regulation of the antiangiogenic factor thrombospondin-1 (TSP-1) in the lung tissue and the serum of the mice. Furthermore, there is a down-regulation of vascular endothelial growth factor (VEGF) in the lung tissue and serum of the B16-F10 tumor-bearing mice treated with PEI-p53 DNA complexes, compared with untreated tumor-bearing animals. In addition, staining for von Willebrand factor (vWF), a marker for the angiogenic blood vessels, revealed that p53 treatment leads to a decrease in the angiogenic phenotype of the B16-F10 tumors. Immunohistochemistry for transgene expression reveals that the PEI-p53 aerosol complexes transfect mainly the epithelial cells lining the airways, with diffuse transfection in the alveolar lining cells, as well as, the tumor foci in the lung tissue. There was also some evidence of apoptosis in the lung tumor foci of animals treated with p53. The data suggest that aerosol delivery of PEI-p53 complexes leads to inhibition of B16-F10 lung metastases, in part by suppression of angiogenesis.
 CT Check Tags: Female
 Administration, Inhalation
 Animals
 Chloramphenicol O-Acetyltransferase: ME, metabolism
 DNA: AD, administration & dosage
 *Drug Delivery Systems
 Endothelial Growth Factors: ME, metabolism
 *Gene Therapy: MT, methods
 *Genes, p53: GE, genetics
 Genetic Vectors
 Humans
 Lung Neoplasms: BS, blood supply
 *Lung Neoplasms: PC, prevention & control
 Lung Neoplasms: SC, secondary
 Lymphokines: ME, metabolism
 Melanoma, Experimental: BS, blood supply
 Melanoma, Experimental: PA, pathology
 *Melanoma, Experimental: PC, prevention & control

Mice

Mice, Inbred C57BL

*Neovascularization, Pathologic: ME, metabolism

Polyethyleneimine: AD, administration & dosage

Thrombospondin 1: ME, metabolism

Transfection

Up-Regulation: PH, physiology

Vascular Endothelial Growth Factor A

Vascular Endothelial Growth Factors

RN 9002-98-6 (Polyethyleneimine); 9007-49-2 (DNA)

CN 0 (Endothelial Growth Factors); 0 (Genetic Vectors); 0 (Lymphokines); 0 (Thrombospondin 1); 0 (Vascular Endothelial Growth Factor A); 0 (Vascular Endothelial Growth Factors); EC 2.3.1.28 (Chloramphenicol O-Acetyltransferase)

L14 ANSWER 2 OF 26 MEDLINE on STN

AN 2002121277 MEDLINE

DN PubMed ID: 11856116

TI Thrombospondin-1, vascular endothelial growth factor expression and their relationship with p53 status in prostate cancer and benign prostatic hyperplasia.

AU Kwak C; Jin R J; Lee C; Park M S; Lee S E

CS Department of Urology and Clinical Research Institute, Seoul National University College of Medicine, Seoul, Korea.

SO BJU international, (2002 Feb) 89 (3) 303-9.

Journal code: 100886721. ISSN: 1464-4096.

CY England: United Kingdom

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals

EM 200203

ED Entered STN: 20020222

Last Updated on STN: 20020324

Entered Medline: 20020322

AB OBJECTIVE: To evaluate the expression of thrombospondin-1 (TSP-1, a potent inhibitor of angiogenesis) and vascular endothelial growth factor (VEGF, an important angiogenic factor in solid tumours) in prostate cancer, and their relationship with p53 status. PATIENTS AND METHODS: Using immunohistochemistry, the expression of VEGF, TSP-1 and p53 was assessed in 82 archival tissue specimens from 23 patients with benign prostatic hyperplasia (BPH), 22 with localized prostate cancer and 37 with metastatic prostate cancer. Seven of the last group had received androgen deprivation therapy. The relationship between the expression of VEGF, TSP-1 and p53 status was also evaluated with tumour grade and stage in patients with prostate cancer. RESULTS: The seven patients receiving hormonal treatment were excluded from the analysis because androgen deprivation significantly increased TSP-1 and decreased VEGF expression (both $P < 0.01$). Immunohistochemical analysis showed significantly higher VEGF and significantly lower TSP-1 expression (both $P < 0.01$) in prostate cancer than in BPH tissues. There was also significantly higher VEGF and significantly lower TSP-1 expression (both $P < 0.05$) in tissues from metastatic than localized prostate cancer. There was no significant correlation between VEGF or TSP-1 expression and Gleason score, but a significant inverse correlation between TSP-1 and VEGF expression. There was a significant association between VEGF expression and p53 status ($P < 0.05$), but TSP-1 expression was not associated with p53 status. CONCLUSIONS: Angiogenic factors, including VEGF and TSP-1, might be important in the development and progression of prostate cancer. These changes seem to be influenced by p53 status. Identifying the angiogenic factors involved in prostate cancer might lead to the development of diagnostic or therapeutic strategies based on

anti-angiogenesis.

CT Check Tags: Male
 Adenocarcinoma: BS, blood supply
 *Adenocarcinoma: ME, metabolism
 Aged
 Aged, 80 and over
 Disease Progression
 *Endothelial Growth Factors: ME, metabolism
 Humans
 Immunohistochemistry
 *Lymphokines: ME, metabolism
 Middle Aged
 Neovascularization, Pathologic
 *Prostatic Hyperplasia: ME, metabolism
 Prostatic Neoplasms: BS, blood supply
 *Prostatic Neoplasms: ME, metabolism
 *Protein p53: ME, metabolism
 Research Support, Non-U.S. Gov't
 *Thrombospondin 1: ME, metabolism
 Vascular Endothelial Growth Factor A
 Vascular Endothelial Growth Factors

CN 0 (Endothelial Growth Factors); 0 (Lymphokines); 0 (Protein p53
); 0 (Thrombospondin 1); 0 (Vascular Endothelial
 Growth Factor A); 0 (Vascular Endothelial Growth Factors)

L14 ANSWER 3 OF 26 MEDLINE on STN

AN 2002071060 MEDLINE

DN PubMed ID: 11796289

TI Thrombospondin-1 expression in patients with
 pathologic stage T3 prostate cancer undergoing radical
 prostatectomy: association with p53 alterations, tumor
 angiogenesis, and tumor progression.

AU Grossfeld Gary D; Carroll Peter R; Lindeman Neil; Meng Maxwell; Groshen
 Susan; Feng An Chen; Hawes Debra; Cote Richard J

CS Department of Urology, University of California, San Francisco, School of
 Medicine, San Francisco, California 94115-1711, USA.

SO Urology, (2002 Jan) 59 (1) 97-102.
 Journal code: 0366151. ISSN: 1527-9995.

CY United States

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals

EM 200202

ED Entered STN: 20020125
 Last Updated on STN: 20020213
 Entered Medline: 20020212

AB OBJECTIVES: To investigate thrombospondin-1 (TSP)
 expression in patients with prostate cancer undergoing
 radical prostatectomy. TSP is a p53-dependent inhibitor of
 tumor angiogenesis. Previous studies have demonstrated that TSP
 expression is significantly associated with the microvessel density (MVD)
 count, p53 expression, and disease-specific and overall survival
 in patients with invasive bladder cancer undergoing radical cystectomy.
 METHODS: Radical prostatectomy specimens from 85 patients with pathologic
 Stage T3 disease were analyzed for TSP expression, p53 nuclear
 reactivity, and MVD using antigen-retrieval immunohistochemistry. The
 median follow-up after surgery was 10.6 years (range 1.8 to 15.4).
 Disease recurrence was defined as a prostate-specific antigen level of 0.2
 ng/mL or greater on two consecutive occasions after surgery. TSP
 expression was graded as present or absent on the basis of the
 immunoreactivity in the extracellular matrix by persons unaware of the
 clinical outcome. Specimens were considered p53 positive
 (altered) if more than 10% of the tumor cells demonstrated nuclear
 reactivity. The chi-square test was used to determine whether the

associations were significant between the pathologic tumor characteristics and the immunohistochemical findings. The log-rank test was used to determine the associations between the immunohistochemical findings and disease recurrence. RESULTS: TSP and p53 were graded as positive in 21 (26%) and 16 (19%) tumors, respectively. The median MVD count was 111.5. No significant associations were found among p53 status, TSP expression, and MVD. Seminal vesicle invasion and Gleason pattern 4 or 5 disease were significant predictors of disease recurrence. A trend was noted toward a higher rate of disease recurrence for patients with altered p53 expression (p53 positive) or increased MVD. TSP expression was not associated with disease recurrence. CONCLUSIONS: We found no significant association between TSP expression and p53 status, MVD count, or outcome after radical prostatectomy for patients with pathologic Stage T3 prostate cancer. Our data suggest that p53 and MVD may be associated with outcome in these patients. Additional studies are needed to identify reliable molecular markers of outcome for patients with this disease.

CT Check Tags: Male

*Adenocarcinoma: CH, chemistry
 Adenocarcinoma: PA, pathology
 Adenocarcinoma: SU, surgery
 Follow-Up Studies
 Humans
 Middle Aged
 Neoplasm Recurrence, Local: BL, blood
 Neoplasm Recurrence, Local: DI, diagnosis
 Neoplasm Staging
 Prostate-Specific Antigen: BL, blood
 Prostatectomy
 *Prostatic Neoplasms: CH, chemistry
 Prostatic Neoplasms: PA, pathology
 Prostatic Neoplasms: SU, surgery
 *Protein p53: AN, analysis
 *Thrombospondin 1: AN, analysis
 *Tumor Markers, Biological: AN, analysis

CN 0 (Protein p53); 0 (Thrombospondin 1); 0
 (Tumor Markers, Biological); EC 3.4.21.77 (Prostate-Specific Antigen)

L14 ANSWER 4 OF 26 MEDLINE on STN

AN 2001155324 MEDLINE

DN PubMed ID: 11205922

TI Independent association of angiogenesis index with outcome in prostate cancer.

AU Mehta R; Kyshtoobayeva A; Kurosaki T; Small E J; Kim H; Stroup R; McLaren C E; Li K T; Fruehauf J P

CS Oncotech Incorporated, Irvine, California 92614, USA.

SO Clinical cancer research : an official journal of the American Association for Cancer Research, (2001 Jan) 7 (1) 81-8.
 Journal code: 9502500. ISSN: 1078-0432.

CY United States

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals

EM 200103

ED Entered STN: 20010404

Last Updated on STN: 20010404

Entered Medline: 20010322

AB New molecular factors have been characterized that are associated with the prognosis of prostate carcinoma patients, including p53 status and angiogenesis. We reported recently that mutant p53 (mp53) was associated with decreased expression of an endogenous inhibitor of angiogenesis, thrombospondin-1 (TSP-1), and increased microvessel density in melanoma and breast

cancer. In this study, we performed a similar analysis on primary prostate carcinoma to determine whether these factors were associated with each other or patient outcomes. Paraffin-embedded specimens of 98 cases of primary prostate carcinoma were obtained and examined to confirm tissue diagnosis and Gleason scores. Carcinoma-specific levels of p53, TSP-1, and tumor angiogenesis were determined using semiquantitative immunohistochemistry (IHC) methods. Acquisition of mp53 was significantly associated with decreased TSP-1 ($P = 0.002$) and increased angiogenesis ($P < 0.0001$). An angiogenesis index integrating mp53, TSP-1, and angiogenesis (CD31) scores was found to be an independent predictor of survival in univariate and multivariate analyses that included Gleason score, clinical stage, and patient age. Further validation of the angiogenesis index in prostate carcinoma may provide a new tool to stratify patient risk.

CT Check Tags: Male
 *Adenocarcinoma: BS, blood supply
 Adenocarcinoma: ME, metabolism
 Adenocarcinoma: SU, surgery
 Aged
 Antigens, CD31: ME, metabolism
 Biopsy, Needle
 Disease Progression
 Humans
 Image Processing, Computer-Assisted
 Immunoenzyme Techniques
 Mutation
 Neovascularization, Pathologic: ME, metabolism
 *Neovascularization, Pathologic: PA, pathology
 Neovascularization, Pathologic: SU, surgery
 Paraffin Embedding
 Prostatectomy
 *Prostatic Neoplasms: BS, blood supply
 Prostatic Neoplasms: ME, metabolism
 Prostatic Neoplasms: SU, surgery
 Protein p53: ME, metabolism
 Retrospective Studies
 Survival Analysis
 Thrombospondin 1: ME, metabolism
 Tumor Markers, Biological: ME, metabolism
 CN 0 (Antigens, CD31); 0 (Protein p53); 0 (Thrombospondin 1); 0 (Tumor Markers, Biological)

L14 ANSWER 5 OF 26 MEDLINE on STN
 AN 2001118033 MEDLINE
 DN PubMed ID: 11150912
 TI Thrombospondin-1 and -2 in node-negative breast cancer: correlation with angiogenic factors, p53, cathepsin D, hormone receptors and prognosis.
 AU Gasparini G; Toi M; Biganzoli E; Dittadi R; Fanelli M; Morabito A; Boracchi P; Gion M
 CS Division of Medical Oncology, Azienda Complesso Ospedaliero 'San Filippo Neri', Rome, Italy.
 SO Oncology, (2001) 60 (1) 72-80.
 Journal code: 0135054. ISSN: 0030-2414.
 CY Switzerland
 DT Journal; Article; (JOURNAL ARTICLE)
 LA English
 FS Priority Journals
 EM 200102
 ED Entered STN: 20010322
 Last Updated on STN: 20010322
 Entered Medline: 20010215
 AB OBJECTIVE: Thrombospondins (TSP(s)) are a multigene family of five secreted glycoproteins involved in the regulation of cell proliferation,

adhesion and migration. Two members of the TSP family, namely TSP-1 and TSP-2, are also naturally occurring inhibitors of angiogenesis. The aim of the present study was to determine the prognostic significance of the determination of TSP-1 and -2 and their correlation with the angiogenic peptides vascular endothelial growth factor (VEGF) and thymidine phosphorylase (TP), as well as with other biological and clinicopathological features investigated. METHODS: We evaluated a series of 168 women with node-negative breast cancer with a median follow-up period of 66 months, not treated with adjuvant therapy. The cytosolic levels of TSP-1 and -2 were determined in the primary tumour by a commercially available immunometric assay. RESULTS: We found that 166 tested tumours had measurable levels of TSP-1 and -2 protein (median value 5.978, range 0.579-31.410 ng/mg of protein). On the basis of Spearman's rank correlation coefficient, a weak inverse association of TSP-1 and -2 with tumour size and cathepsin D was found. Moreover, principal component analysis on ranks evidenced a poor association between TSP-1 and -2, VEGF and TP. The results of the clinical outcome were analysed by both univariate and multivariate [for relapse-free survival (RFS) only] Cox regression models. TSP-1 and -2 were not significant prognostic factors in univariate analysis for either RFS ($p = 0.427$) or overall survival ($p = 0.069$). To investigate the 'angiogenic balance hypothesis', bivariate analyses were performed to investigate the interactions of TSP-1 and -2 with VEGF, TP or p53, but none were included in the selected models. Finally, in multivariate analysis for RFS a baseline model, previously defined in a larger case series and inclusive of VEGF, TP and their interaction was adopted. It was highly significant ($p = 0.002$, Harrell c statistic value of 0.703); but when TSP-1 and -2 were added, their contribution was negligible ($p = 0.731$, Harrell c statistic value of 0.705). CONCLUSIONS: The results of this study suggest that TSP-1 and -2 do not provide additional prognostic contribution to the joint effects of VEGF and TP. In the series of node-negative breast cancer patients investigated, determination of the angiogenic peptides VEGF and TP gave significant prognostic information. On the contrary, TSP-1 and -2, potential naturally occurring negative regulators of angiogenesis, lacked prognostic value.

CT Check Tags: Female

- *Breast Neoplasms: CH, chemistry
- Breast Neoplasms: PA, pathology
- *Cathepsin D: AN, analysis
- Cytosol: CH, chemistry
- *Endothelial Growth Factors: AN, analysis
- Humans
- Immunohistochemistry
- *Lymphokines: AN, analysis
- Neovascularization, Pathologic: ME, metabolism
- Predictive Value of Tests
- Prognosis
- Proportional Hazards Models
- *Protein p53: AN, analysis
- *Receptors, Estrogen: AN, analysis
- *Receptors, Progesterone: AN, analysis
- Research Support, Non-U.S. Gov't
- Thrombospondin 1: AN, analysis
- *Thrombospondins: AN, analysis
- Thymidine Phosphorylase: AN, analysis
- *Tumor Markers, Biological: AN, analysis
- Vascular Endothelial Growth Factor A
- Vascular Endothelial Growth Factors

CN 0 (Endothelial Growth Factors); 0 (Lymphokines); 0 (Protein p53); 0 (Receptors, Estrogen); 0 (Receptors, Progesterone); 0 (Thrombospondin 1); 0 (Thrombospondins); 0 (Tumor Markers, Biological); 0 (Vascular Endothelial Growth Factor A); 0 (Vascular Endothelial Growth Factors); EC 2.4.2.4 (Thymidine

Phosphorylase); EC 3.4.23.5 (Cathepsin D)

L14 ANSWER 6 OF 26 MEDLINE on STN
AN 2000182650 MEDLINE
DN PubMed ID: 10719731
TI p53 and vascular-endothelial-growth-factor (VEGF) expression predicts outcome in 833 patients with primary breast carcinoma.
AU Linderholm B; Lindh B; Tavelin B; Grankvist K; Henriksson R
CS Department of Oncology, Umea University, Sweden..
Barbro.Linderholm@onkologi.umu.se
SO International journal of cancer. Journal international du cancer, (2000 Jan 20) 89 (1) 51-62.
Journal code: 0042124. ISSN: 0020-7136.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200003
ED Entered STN: 20000330
Last Updated on STN: 20000330
Entered Medline: 20000321
AB The angiogenic factor vascular endothelial growth factor (VEGF) predicts outcome in primary breast carcinoma. Alteration of the p53 gene causes down-regulation of the expression of thrombospondin-1, a natural inhibitor of angiogenesis. This study was conducted to investigate the association between mutant p53 protein and VEGF expression, and the prognostic value of these factors. VEGF165 and p53 protein were measured in tumour cytosols by enzyme immunoassays. Recurrence-free survival (RFS) and overall survival (OS) were estimated in 833 consecutive patients, 485 node-negative (NNBC) and 348 node-positive (NPBC) with primary invasive breast cancer. A significant association was found between mutant p53 protein and VEGF expression. Univariate analysis showed both p53 and VEGF to be significant predictors of survival. Similar correlation was seen when p53 was combined with VEGF. Univariate analysis of NNBC showed significant prognostic value of p53 for OS, also when combined with VEGF expression; for NPBC, significant reductions in RFS and OS were seen for p53-positive patients, and these findings were enhanced when combined with VEGF, also in the sub-group receiving adjuvant endocrine treatment. Multivariate analysis showed both p53 and VEGF as independent predictors of OS in all groups. When the 2 factors were combined, an increased relative risk of 2.7 was seen for OS in the group with both p53 positivity and high VEGF content, as compared with 1.7 in the group with one risk factor. The results suggest an association between loss of wt-p53 and increased VEGF expression, indicating that angiogenic activity may depend, at least partly, on altered p53-protein function. Combination of these 2 biological markers appears to give additional predictive information of survival. A high-risk group of patients was associated with p53 positivity and higher VEGF content.
CT Check Tags: Female
Breast Neoplasms: BS, blood supply
*Breast Neoplasms: ME, metabolism
Breast Neoplasms: MO, mortality
Breast Neoplasms: PA, pathology
*Endothelial Growth Factors: ME, metabolism
Humans
*Lymphokines: ME, metabolism
Multivariate Analysis
Neovascularization, Pathologic
Prognosis
Proportional Hazards Models
*Protein p53: ME, metabolism

Receptors, Estrogen: ME, metabolism
 Receptors, Progesterone: ME, metabolism
 Research Support, Non-U.S. Gov't
 Survival Analysis
 Vascular Endothelial Growth Factor A
 Vascular Endothelial Growth Factors
 CN 0 (Endothelial Growth Factors); 0 (Lymphokines); 0 (Protein p53
); 0 (Receptors, Estrogen); 0 (Receptors, Progesterone); 0 (Vascular
 Endothelial Growth Factor A); 0 (Vascular Endothelial Growth Factors); 0
 (vascular endothelial growth factor A, human)

L14 ANSWER 7 OF 26 MEDLINE on STN
 AN 1999240722 MEDLINE
 DN PubMed ID: 10224095
 TI Systemic gene delivery expands the repertoire of effective antiangiogenic
 agents.
 AU Liu Y; Thor A; Shtivelman E; Cao Y; Tu G; Heath T D; Debs R J
 CS Geraldine Brush Cancer Research Institute at the California Pacific
 Medical Center, San Francisco, California 94115, USA.
 NC CA58207 (NCI)
 CA58914 (NCI)
 CA71422 (NCI)
 SO Journal of biological chemistry, (1999 May 7) 274 (19) 13338-44.
 Journal code: 2985121R. ISSN: 0021-9258.
 CY United States
 DT Journal; Article; (JOURNAL ARTICLE)
 LA English
 FS Priority Journals
 EM 199906
 ED Entered STN: 19990614
 Last Updated on STN: 19990614
 Entered Medline: 19990603

AB Cationic liposome-DNA complex (CLDC)-based intravenous gene delivery
 targets gene expression to vascular endothelial cells, macrophages and
 tumor cells. We used systemic gene delivery to identify anti-angiogenic
 gene products effective against metastatic spread in tumor-bearing mice.
 Specifically, CLDC-based intravenous delivery of the p53 and
 GM-CSF genes were each as effective as the potent antiangiogenic gene,
 angiostatin, in reducing both tumor metastasis and tumor
 angiogenesis. Combined delivery of these genes did not increase
 anti-tumor activity, further suggesting that each gene appeared to produce
 its antimetastatic activity through a common antiangiogenic pathway.
 CLDC-based intravenous delivery of the human wild type p53 gene
 transfected up to 80% of tumor cells metastatic to lung. Furthermore, it
 specifically induced the expression of the potent antiangiogenic gene,
 thrombospondin-1, indicating that p53 gene
 delivery in vivo may inhibit angiogenesis by inducing endogenous
 thrombospondin-1 expression. CLDC-based delivery also
 identified a novel anti-tumor activity for the metastasis suppressor gene
 CC3. Thus, CLDC-based intravenous gene delivery can produce systemic
 antiangiogenic gene therapy using a variety of different genes and may be
 used to assess potential synergy of combined anti-tumor gene delivery and
 to identify novel activities for existing anti-tumor genes.

CT Angiostatins
 Animals
 Gene Expression
 *Gene Transfer Techniques
 Genes, p53: GE, genetics
 Granulocyte-Macrophage Colony-Stimulating Factor: GE, genetics
 Humans
 *Melanoma, Experimental: BS, blood supply
 Melanoma, Experimental: GE, genetics
 Melanoma, Experimental: PA, pathology
 Mice

*Neoplasm Metastasis: TH, therapy
Neovascularization, Pathologic: GE, genetics
*Neovascularization, Pathologic: TH, therapy
Peptide Fragments: GE, genetics
Plasminogen: GE, genetics
Research Support, Non-U.S. Gov't
Research Support, U.S. Gov't, P.H.S.

Thrombospondin 1: GE, genetics

RN 83869-56-1 (Granulocyte-Macrophage Colony-Stimulating Factor); 86090-08-6
(Angiostatins); 9001-91-6 (Plasminogen)
CN 0 (Peptide Fragments); 0 (Thrombospondin 1)

L14 ANSWER 8 OF 26 MEDLINE on STN

AN 1998278601 MEDLINE

DN PubMed ID: 9618039

TI Mutant p53 correlates with reduced expression of
thrombospondin-1, increased angiogenesis, and
metastatic progression in melanoma.

AU Grant S W; Kyshtoobayeva A S; Kurosaki T; Jakowatz J; Fruehauf J P
CS Department of Surgery, University of California, Irvine College of
Medicine, USA.

SO Cancer detection and prevention, (1998) 22 (3) 185-94.
Journal code: 7704778. ISSN: 0361-090X.

CY United States

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals

EM 199902

ED Entered STN: 19990316

Last Updated on STN: 19990316

Entered Medline: 19990226

AB On the basis of reports linking mutant p53 (mp53) to decreased
expression of the angiogenesis inhibitor thrombospondin
-1 (TSP-1) and increased angiogenesis, we compared
primary and metastatic melanoma tumor specimens to determine if
these factors were associated with metastatic progression. Western
blotting, immunohistochemistry (IHC), and image analysis (IA) techniques
were employed to evaluate the relationship between p53 status
and TSP-1 expression in Zaz and M14 melanoma cell lines, and
among p53, TSP-1, and angiogenesis in primary and
metastatic melanomas. Zaz cells expressed wild-type p53
(WT p53) and high levels of TSP-1, while the M14 cells expressed
mp53 and low TSP-1 levels. Examination of clinical melanoma
specimens (N = 99) revealed an incidence of mp53 of 48%. Specimens with
WT p53 (N = 46) expressed significantly higher mean levels of
TSP-1 (41 +/- 27 vs. 21 +/- 24; p = 0.0004), and lower microvessel counts
per 200x field (25 +/- 17 vs. 40 +/- 20; p = 0.0001) than tumors
expressing mp53 (N = 42). A significantly higher incidence of mp53
expression was seen in metastatic tumors (64%, 37/58) than in primary
tumors (27%, 11/41) (p < 0.0005). Primary tumors specimens had higher
levels of TSP-1 (40 +/- 27 vs. 25 +/- 25; p = 0.0054) and lower
microvessel counts (26 +/- 18 vs. 39 +/- 20, p = 0.0013) than metastatic
tumors. These data suggest that acquisition of mp53, decreased TSP-1, and
increased microvessel infiltration may be interrelated and associated with
the metastatic phenotype in malignant melanoma.

CT Blotting, Western

*Genes, p53: GE, genetics

Humans

Immunohistochemistry

Melanoma: BS, blood supply

*Melanoma: GE, genetics

*Melanoma: SC, secondary

*Mutation: GE, genetics

*Neovascularization, Pathologic: GE, genetics

Thrombospondins: AI, antagonists & inhibitors
*Thrombospondins: BI, biosynthesis
Tumor Cells, Cultured

CN 0 (Thrombospondins)

L14 ANSWER 9 OF 26 MEDLINE on STN

AN 96049797 MEDLINE

DN PubMed ID: 8534861

TI The modulation of thrombospondin and other naturally occurring inhibitors of angiogenesis during tumor progression.

AU Volpert O V; Stellmach V; Bouck N

CS Department of Microbiology-Immunology, Northwestern University, Chicago, IL 60611, USA.

NC RO1 CA27350 (NCI)

SO Breast cancer research and treatment, (1995) 36 (2) 119-26. Ref: 56
Journal code: 8111104. ISSN: 0167-6806.

CY Netherlands

DT Journal; Article; (JOURNAL ARTICLE)

General Review; (REVIEW)

(REVIEW, TUTORIAL)

LA English

FS Priority Journals

EM 199602

ED Entered STN: 19960221

Last Updated on STN: 19980206

Entered Medline: 19960207

AB Fifteen different natural inhibitors of angiogenesis have now been identified that are produced by mammalian cells and are able to block in vivo neovascularization. The majority of these are able to inhibit endothelial cell activities in vitro and all those tested have demonstrated significant antitumor activity. Most normal cells produce inhibitors of neovascularization that must be downregulated before the cells can develop into angiogenic, malignant tumors. In several cases the production of inhibitors ceases when tumor suppressor genes are inactivated. In the BT549 human breast carcinoma cell line, the reintroduction of a wild type p53 tumor suppressor gene resulted in the stimulation of the secretion of an inhibitor of angiogenesis, thrombospondin-1, and as a result the cells lost their angiogenic phenotype and became able to suppress angiogenesis induced by the parental tumor line. These results provide a new example of tumor suppressor gene control of a natural inhibitor of angiogenesis and add support to the concept that thrombospondin loss may play an important role in the development of some human breast cancers.

CT Animals

*Breast Neoplasms: BS, blood supply

*Breast Neoplasms: ME, metabolism

Cell Adhesion Molecules: BI, biosynthesis

Cell Adhesion Molecules: ME, metabolism

Disease Progression

Down-Regulation

Humans

*Membrane Glycoproteins: BI, biosynthesis

Membrane Glycoproteins: PH, physiology

*Neovascularization, Pathologic: ME, metabolism

Research Support, Non-U.S. Gov't

Research Support, U.S. Gov't, P.H.S.

Thrombospondins

CN 0 (Cell Adhesion Molecules); 0 (Membrane Glycoproteins); 0
(Thrombospondins)

L14 ANSWER 10 OF 26 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on
STN

AN 2001:85345 BIOSIS

DN PREV200100085345
 TI **Angiogenesis** index (AI) is associated with early recurrence in patients presenting with primary **breast cancer**.
 AU Ellis, R. J. [Reprint author]; Kimler, B. F. [Reprint author]; Fabian, C. J. [Reprint author]; Tawfik, O. [Reprint author]; Mehta, R. S.; Kysthoobayeva, A.; Fruehauf, J. P.
 CS University of Kansas Medical Center, Kansas City, KS, USA
 SO Breast Cancer Research and Treatment, (November, 2000) Vol. 64, No. 1, pp. 101. print.
 Meeting Info.: 23rd Annual San Antonio Breast Cancer Symposium. San Antonio, Texas, USA. December 06-09, 2000. Cancer Therapy and Research Center Research Foundation.
 CODEN: BCTRD6. ISSN: 0167-6806.
 DT Conference; (Meeting)
 Conference; Abstract; (Meeting Abstract)
 Conference; (Meeting Poster)
 LA English
 ED Entered STN: 14 Feb 2001
 Last Updated on STN: 12 Feb 2002
 CC Immunology - General and methods 34502
 General biology - Symposia, transactions and proceedings 00520
 Biochemistry studies - Proteins, peptides and amino acids 10064
 Biochemistry studies - Sterols and steroids 10067
 Cardiovascular system - Physiology and biochemistry 14504
 Blood - Blood and lymph studies 15002
 Blood - Blood cell studies 15004
 Reproductive system - Physiology and biochemistry 16504
 Reproductive system - Pathology 16506
 Endocrine - General 17002
 Neoplasms - Immunology 24003
 Neoplasms - Pathology, clinical aspects and systemic effects 24004
 Immunology - Immunopathology, tissue immunology 34508
 IT Major Concepts
 Gynecology (Human Medicine, Medical Sciences); Oncology (Human Medicine, Medical Sciences); Methods and Techniques
 IT Parts, Structures, & Systems of Organisms
 blood vessel: circulatory system; breast: reproductive system, histology; lymph node: blood and lymphatics, immune system, histology
 IT Diseases
 primary **breast cancer**: neoplastic disease, reproductive system disease/female, early recurrence, grade, invasiveness
 Breast Neoplasms (MeSH)
 IT Chemicals & Biochemicals
 CD31: biomarker, expression; estrogen; estrogen receptor: expression; p53: biomarker, expression; progesterone; progesterone receptor: expression; **thrombospondin-1** [TSP-1]: biomarker, expression
 IT Methods & Equipment
angiogenesis index: scoring method
 IT Miscellaneous Descriptors
 age; **angiogenesis**; blood vessel density; estrogen receptor status; invasive phenotype; lymph node status; progesterone receptor status; survival rate; tumor grade; tumor size; Meeting Abstract; Meeting Poster
 ORGN Classifier
 Hominidae 86215
 Super Taxa
 Primates; Mammalia; Vertebrata; Chordata; Animalia
 Organism Name
 human: female, patient
 Taxa Notes
 Animals, Chordates, Humans, Mammals, Primates, Vertebrates
 RN 57-83-0 (progesterone)

L14 ANSWER 11 OF 26 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on
STN

AN 2000:238815 BIOSIS

DN PREV200000238815

TI Importance of vascular endothelial growth factor (VEGF) and
thrombospondin-1 (TSP-1) in melanoma
angiogenesis, and independent prognostic significance of
microvessel density.

AU Straume, Oddbjorn [Reprint author]; Akslen, Lars A. [Reprint author]

CS Gade Institute, Bergen, Norway

SO Proceedings of the American Association for Cancer Research Annual
Meeting, (March, 2000) No. 41, pp. 511. print.
Meeting Info.: 91st Annual Meeting of the American Association for Cancer
Research. San Francisco, California, USA. April 01-05, 2000.
ISSN: 0197-016X.

DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)

LA English

ED Entered STN: 7 Jun 2000
Last Updated on STN: 5 Jan 2002

CC Neoplasms - General 24002
Biochemistry studies - General 10060
Cardiovascular system - General and methods 14501
General biology - Symposia, transactions and proceedings 00520

IT Major Concepts
Cardiovascular System (Transport and Circulation); Tumor Biology

IT Parts, Structures, & Systems of Organisms
microvessels: circulatory system, density

IT Diseases
melanoma: neoplastic disease
Melanoma (MeSH)

IT Chemicals & Biochemicals
Ki-67: expression; p16 protein: expression; p53: expression;
thrombospondin-1: expression; vascular endothelial
growth factor: expression

IT Methods & Equipment
immunohistochemistry: analytical method; in situ hybridization:
analytical method

IT Miscellaneous Descriptors
angiogenesis; disease prognosis; disease survival; tumor
stage; Meeting Abstract

ORGN Classifier
Hominidae 86215
Super Taxa
Primates; Mammalia; Vertebrata; Chordata; Animalia
Organism Name
human: patient
Taxa Notes
Animals, Chordates, Humans, Mammals, Primates, Vertebrates

RN 127464-60-2 (vascular endothelial growth factor)

L14 ANSWER 12 OF 26 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on
STN

AN 1998:280331 BIOSIS

DN PREV199800280331

TI p53 and angiogenesis in neoplasia.

AU Gasparini, Giampietro [Reprint author]; Harris, Adrian L.

CS Dep. Oncology, St. Bortolo Hosp., 36100 Vicenza, Italy

SO Klijn, J. G. M. [Editor]. (1997) pp. 115-130. European School of Oncology
Scientific Updates, Vol. 1; Prognostic and predictive value of p53. print.
Publisher: Elsevier Science Publishers B.V., PO Box 211, Sara
Burgerhartstraat 25, 1000 AE Amsterdam, The Netherlands; Elsevier Science
Publishing Co., Inc., P.O. Box 882, Madison Square Station, New York, New

York 10159-2101, USA.
 ISBN: 0-444-82832-X.
 DT Book
 Book; (Book Chapter)
 LA English
 ED Entered STN: 8 Jul 1998
 Last Updated on STN: 8 Jul 1998
 CC Genetics - General 03502
 Biochemistry studies - General 10060
 Metabolism - General metabolism and metabolic pathways 13002
 Cardiovascular system - General and methods 14501
 Neoplasms - General 24002
 IT Major Concepts
 Cardiovascular System (Transport and Circulation); Molecular Genetics
 (Biochemistry and Molecular Biophysics); Tumor Biology
 IT Diseases
 breast cancer: neoplastic disease, reproductive
 system disease/female
 Breast Neoplasms (MeSH)
 IT Chemicals & Biochemicals
 p53: inactivation, mutation, tumor suppressor gene;
 thrombospondin-1
 IT Miscellaneous Descriptors
 angiogenesis; Book Chapter
 ORGN Classifier
 Hominidae 86215
 Super Taxa
 Primates; Mammalia; Vertebrata; Chordata; Animalia
 Organism Name
 human: patient
 Taxa Notes
 Animals, Chordates, Humans, Mammals, Primates, Vertebrates
 L14 ANSWER 13 OF 26 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on
 STN
 AN 1998:194893 BIOSIS
 DN PREV199800194893
 TI Regulation of angiogenesis in carcinoma of the breast, prostate,
 colon, and malignant melanoma by p53 and
 thrombospondin-1 (TSP1).
 AU Fruehauf, J. P. [Reprint author]; Mehta, R.; Mechetner, E.; Kurosaki, T.;
 Jackowatz, J.; Grant, S.; Kyshtoobayeva, A.
 CS Oncotech Inc., Irvine, CA 92614, USA
 SO Proceedings of the American Association for Cancer Research Annual
 Meeting, (March, 1998) Vol. 39, pp. 150. print.
 Meeting Info.: 89th Annual Meeting of the American Association for Cancer
 Research. New Orleans, Louisiana, USA. March 28-April 1, 1998. American
 Association for Cancer Research.
 ISSN: 0197-016X.
 DT Conference; (Meeting)
 Conference; Abstract; (Meeting Abstract)
 LA English
 ED Entered STN: 4 May 1998
 Last Updated on STN: 4 May 1998
 CC Neoplasms - Biochemistry 24006
 Cardiovascular system - Physiology and biochemistry 14504
 Reproductive system - Pathology 16506
 General biology - Symposia, transactions and proceedings 00520
 IT Major Concepts
 Cardiovascular System (Transport and Circulation); Cell Biology; Tumor
 Biology
 IT Diseases
 breast carcinoma: neoplastic disease, reproductive system
 disease/female

Breast Neoplasms (MeSH); Carcinoma (MeSH)

IT Diseases
colon carcinoma: digestive system disease, neoplastic disease
Colonic Neoplasms (MeSH); Carcinoma (MeSH)

IT Diseases
malignant melanoma: neoplastic disease
Melanoma (MeSH)

IT Diseases
prostate carcinoma: neoplastic disease, reproductive system
disease/male, urologic disease
Prostatic Neoplasms (MeSH); Carcinoma (MeSH)

IT Chemicals & Biochemicals
p53; thrombospondin-1 [TSP1]

IT Miscellaneous Descriptors
angiogenesis regulation; tumor physiology; Meeting Abstract

L14 ANSWER 14 OF 26 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on
STN

AN 1998:154913 BIOSIS

DN PREV199800154913

TI **Thrombospondin-1 in invasive breast
cancer and its association with p53 expression, micro
vessel density and clinical outcome.**

AU Steward, M. A. [Reprint author]; Rice, A. J.; Roberts, D.; Benson, E. A.;
Horgan, K.; Quinn, C. M.

CS Dep. Surg., Gen. Infirmary at Leeds, Leeds, UK

SO Journal of Pathology, (1998) Vol. 184, No. SUPPL., pp. 5A. print.
Meeting Info.: 176th Meeting of the Pathological Society of Great Britain
and Ireland. London, England, UK. January 7-9, 1998. Departments of
Histopathology and Medical Microbiology, Imperial College School of
Medicine at Charing Cross, London.
CODEN: JPTLAS. ISSN: 0022-3417.

DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)

LA English

ED Entered STN: 31 Mar 1998
Last Updated on STN: 31 Mar 1998

CC Pathology - General 12502
Microscopy - Histology and histochemistry 01056
Biochemistry studies - Proteins, peptides and amino acids 10064
Replication, transcription, translation 10300
Pathology - Diagnostic 12504
Metabolism - Proteins, peptides and amino acids 13012
Cardiovascular system - General and methods 14501
Reproductive system - General and methods 16501
Neoplasms - Pathology, clinical aspects and systemic effects 24004
Neoplasms - Biochemistry 24006
Neoplasms - Carcinogens and carcinogenesis 24007
General biology - Symposia, transactions and proceedings 00520

IT Major Concepts
Reproductive System (Reproduction); Tumor Biology

IT Diseases
breast cancer: neoplastic disease, reproductive
system disease/female
Breast Neoplasms (MeSH)

IT Diseases
invasive breast cancer: neoplastic disease,
reproductive system disease/female
Breast Neoplasms (MeSH)

IT Chemicals & Biochemicals
p53: expression; thrombospondin-1

IT Methods & Equipment
immunohistochemistry: analytical method

IT Miscellaneous Descriptors

angiogenesis; clinical outcome; micro vessel density; tumor
 grades; Meeting Abstract
 ORGN Classifier
 Hominidae 86215
 Super Taxa
 Primates; Mammalia; Vertebrata; Chordata; Animalia
 Organism Name
 human: female, patient
 Taxa Notes
 Animals, Chordates, Humans, Mammals, Primates, Vertebrates

L14 ANSWER 15 OF 26 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on
 STN
 AN 1997:422735 BIOSIS
 DN PREV199799721938
 TI Control of inhibitors of angiogenesis by tumor suppressor genes.
 AU Bouck, Noel
 CS Northwest. Univ. Med. Sch., Chicago, IL, USA
 SO FASEB Journal, (1997) Vol. 11, No. 9, pp. A1450.
 Meeting Info.: 17th International Congress of Biochemistry and Molecular
 Biology in conjunction with the Annual Meeting of the American Society for
 Biochemistry and Molecular Biology. San Francisco, California, USA. August
 24-29, 1997.
 CODEN: FAJOEC. ISSN: 0892-6638.

DT Conference; (Meeting)
 Conference; Abstract; (Meeting Abstract)

LA English
 ED Entered STN: 8 Oct 1997
 Last Updated on STN: 8 Oct 1997

CC General biology - Symposia, transactions and proceedings 00520
 Genetics - Animal 03506
 Biochemistry studies - Proteins, peptides and amino acids 10064
 Biophysics - Membrane phenomena 10508
 Cardiovascular system - Blood vessel pathology 14508
 Respiratory system - Pathology 16006
 Endocrine - General 17002
 Neoplasms - Biochemistry 24006
 Neoplasms - Carcinogens and carcinogenesis 24007

IT Major Concepts
 Biochemistry and Molecular Biophysics; Cardiovascular Medicine (Human
 Medicine, Medical Sciences); Endocrine System (Chemical Coordination
 and Homeostasis); Genetics; Membranes (Cell Biology); Oncology (Human
 Medicine, Medical Sciences); Pulmonary Medicine (Human Medicine,
 Medical Sciences)

IT Miscellaneous Descriptors
 ANGIOGENESIS; BASIC FIBROBLAST GROWTH FACTOR; BFGF; CD36;
 FIBROSARCOMA; LUNG METASTASIS; MELANOMA; MICROVASCULAR CELL;
 MIGRATION; MOLECULAR GENETICS; NEOPLASTIC DISEASE; P53;
 RESPIRATORY SYSTEM DISEASE; THROMBOSPONDIN-1; TUMOR
 BIOLOGY; VASCULAR ENDOTHELIAL GROWTH FACTOR; VEGF

ORGN Classifier
 Hominidae 86215
 Super Taxa
 Primates; Mammalia; Vertebrata; Chordata; Animalia
 Organism Name
 human
 Taxa Notes
 Animals, Chordates, Humans, Mammals, Primates, Vertebrates

ORGN Classifier
 Muridae 86375
 Super Taxa
 Rodentia; Mammalia; Vertebrata; Chordata; Animalia
 Organism Name
 mouse

Taxa Notes

Animals, Chordates, Mammals, Nonhuman Vertebrates, Nonhuman Mammals,
Rodents, Vertebrates

L14 ANSWER 16 OF 26 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on
STN
AN 1997:249509 BIOSIS
DN PREV199799548712
TI Regulation of genes associated with angiogenesis, growth, and
metastasis by specific p53 point mutations in a murine
melanoma cell line.
AU Koura, Aaryan N.; Van Golen, Kenneth; Tsan, Rachel; Radinsky, Robert;
Price, Janet E.; Ellis, Lee M. [Reprint author]
CS Dep. Surg. Oncol., Box 106, Univ. Texas M.D. Anderson Cancer Cent., 1515
Holcombe Blvd., Houston, TX 77030, USA
SO Oncology Reports, (1997) Vol. 4, No. 3, pp. 475-479.
ISSN: 1021-335X.
DT Article
LA English
ED Entered STN: 13 Jun 1997
Last Updated on STN: 13 Jun 1997
AB K1735 murine melanoma cells transfected with p53 cDNAs
bearing specific point mutations are metastatic in nude mice, whereas the
parent and control-transfected cells are nonmetastatic. To determine
whether p53 gene mutations regulate genes associated with
angiogenesis, growth, and metastasis, we examined expression of
vascular endothelial growth factor, transforming growth factor-beta,
mdm-2, insulin-like growth factor I, IGF-I receptor, epidermal growth
factor receptor, c-MET, and thrombospondin 1 in K1735
cells transfected with one of four different mutant p53 cDNAs.
Northern blot analysis demonstrated differential upregulation of these
genes in cells transfected with different mutant p53 cDNAs.
Up-regulation of angiogenesis-, growth-, and metastasis-related
genes by mutant p53 may contribute to metastasis formation.
CC Genetics - Animal 03506
Biochemistry studies - General 10060
Neoplasms - General 24002
IT Major Concepts
Biochemistry and Molecular Biophysics; Genetics; Tumor Biology
IT Chemicals & Biochemicals
INSULIN-LIKE GROWTH FACTOR I
IT Miscellaneous Descriptors
ANGIOGENESIS; C-MET; EPIDERMAL GROWTH FACTOR RECEPTOR;
EXPRESSION; GENE REGULATION; GENETICS; INSULIN-LIKE GROWTH FACTOR I;
INSULIN-LIKE GROWTH FACTOR I RECEPTOR; K1735 CELL LINE; MDM-2;
METASTASIS; MURINE MELANOMA CELLS; NUDE MOUSE; P53
DNA; P53 POINT MUTATIONS; THROMBOSPONDIN 1
; TRANSFORMING GROWTH FACTOR-BETA; TUMOR BIOLOGY; TUMOR GROWTH;
VASCULAR ENDOTHELIAL GROWTH FACTOR
ORGN Classifier
Muridae 86375
Super Taxa
Rodentia; Mammalia; Vertebrata; Chordata; Animalia
Organism Name
Muridae
Taxa Notes
Animals, Chordates, Mammals, Nonhuman Vertebrates, Nonhuman Mammals,
Rodents, Vertebrates
RN 67763-96-6 (INSULIN-LIKE GROWTH FACTOR I)

L14 ANSWER 17 OF 26 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on
STN
AN 1997:233070 BIOSIS
DN PREV199799532273

TI P53, thrombospondin-1 (TSP-1),
 angiogenesis (ANG) and androgen receptor (AR) as prognostic
 factors in prostate cancer (PC).
 AU Mehta, R. [Reprint author]; Kyshtoobayeva, A.; Kurosaki, T.; Small, E.;
 Stroop, R.; Fruehauf, J.
 CS Oncotech Inc., Irvine, CA 92614, USA
 SO Proceedings of the American Association for Cancer Research Annual
 Meeting, (1997) Vol. 38, No. 0, pp. 429.
 Meeting Info.: Eighty-eighth Annual Meeting of the American Association
 for Cancer Research. San Diego, California, USA. April 12-16, 1997.
 ISSN: 0197-016X.
 DT Conference; (Meeting)
 Conference; Abstract; (Meeting Abstract)
 LA English
 ED Entered STN: 2 Jun 1997
 Last Updated on STN: 2 Jun 1997
 CC General biology - Symposia, transactions and proceedings 00520
 Biophysics - Membrane phenomena 10508
 Metabolism - Carbohydrates 13004
 Metabolism - Proteins, peptides and amino acids 13012
 Cardiovascular system - Blood vessel pathology 14508
 Blood - Blood cell studies 15004
 Urinary system - Pathology 15506
 Reproductive system - Pathology 16506
 Neoplasms - Biochemistry 24006
 IT Major Concepts
 Blood and Lymphatics (Transport and Circulation); Cardiovascular
 Medicine (Human Medicine, Medical Sciences); Membranes (Cell Biology);
 Metabolism; Oncology (Human Medicine, Medical Sciences); Reproductive
 System (Reproduction); Urology (Human Medicine, Medical Sciences)
 IT Miscellaneous Descriptors
 ANDROGEN RECEPTOR; ANGIOGENESIS; EXPRESSION; NEOPLASTIC
 DISEASE; PATIENT; PROGNOSTIC MARKER; PROSTATE CANCER
 ; P53; REPRODUCTIVE SYSTEM DISEASE/MALE; SURVIVAL;
 THROMBOSPONDIN-1; TUMOR BIOLOGY; UROLOGIC DISEASE
 ORGN Classifier
 Hominidae 86215
 Super Taxa
 Primates; Mammalia; Vertebrata; Chordata; Animalia
 Organism Name
 human
 Taxa Notes
 Animals, Chordates, Humans, Mammals, Primates, Vertebrates
 L14 ANSWER 18 OF 26 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on
 STN
 AN 1997:231779 BIOSIS
 DN PREV199799530982
 TI Mutant p53, TSP-1, and angiogenesis: An index of
 metastatic risk in breast cancer.
 AU Fruehauf, J. [Reprint author]; Kyshtoobayeva, A.; Yeatman, T.; Coppola,
 D.; Kurosaki, T.; Kim, H.
 CS Oncotech Inc., Irvine, CA 92614, USA
 SO Proceedings of the American Association for Cancer Research Annual
 Meeting, (1997) Vol. 38, No. 0, pp. 234-235.
 Meeting Info.: Eighty-eighth Annual Meeting of the American Association
 for Cancer Research. San Diego, California, USA. April 12-16, 1997.
 ISSN: 0197-016X.
 DT Conference; (Meeting)
 Conference; Abstract; (Meeting Abstract)
 LA English
 ED Entered STN: 2 Jun 1997
 Last Updated on STN: 2 Jun 1997
 CC General biology - Symposia, transactions and proceedings 00520

Cytology - Human 02508
 Genetics - Human 03508
 Biochemistry studies - Nucleic acids, purines and pyrimidines 10062
 Biochemistry studies - Proteins, peptides and amino acids 10064
 Replication, transcription, translation 10300
 Biophysics - Molecular properties and macromolecules 10506
 Anatomy and Histology - Microscopic and ultramicroscopic anatomy 11108
 Pathology - Diagnostic 12504
 Metabolism - Proteins, peptides and amino acids 13012
 Metabolism - Nucleic acids, purines and pyrimidines 13014
 Cardiovascular system - Physiology and biochemistry 14504
 Cardiovascular system - Blood vessel pathology 14508
 Reproductive system - Anatomy 16502
 Reproductive system - Physiology and biochemistry 16504
 Reproductive system - Pathology 16506
 Neoplasms - Diagnostic methods 24001
 Neoplasms - Immunology 24003
 Neoplasms - Biochemistry 24006
 Neoplasms - Carcinogens and carcinogenesis 24007
 Development and Embryology - Morphogenesis 25508
 Immunology - General and methods 34502

IT Major Concepts

Biochemistry and Molecular Biophysics; Cardiovascular Medicine (Human Medicine, Medical Sciences); Cardiovascular System (Transport and Circulation); Cell Biology; Development; Genetics; Immune System (Chemical Coordination and Homeostasis); Metabolism; Molecular Genetics (Biochemistry and Molecular Biophysics); Morphology; Oncology (Human Medicine, Medical Sciences); Pathology; Reproductive System (Reproduction)

IT Miscellaneous Descriptors

ANGIOGENESIS; BLOOD VESSEL FORMATION INHIBITOR;
 BREAST CANCER; DIAGNOSTIC METHOD; EXPRESSION; FEMALE;
 GENETIC DISEASE; IMMUNOHISTOCHEMISTRY; IMMUNOLOGIC METHOD; MEDICAL GENETICS; METASTASIS; METASTATIC RISK; MOLECULAR BIOLOGY; MUTANT P53; MUTATION; NEOPLASTIC DISEASE; ONCOLOGY; PATIENT; P53 TUMOR SUPPRESSOR GENE; REPRODUCTIVE SYSTEM DISEASE/FEMALE; SURVIVAL; THROMBOSPONDIN-1; TSP-1; TUMOR PROGRESSION

ORGN Classifier

Hominidae 86215
 Super Taxa
 Primates; Mammalia; Vertebrata; Chordata; Animalia
 Organism Name
 human
 Taxa Notes
 Animals, Chordates, Humans, Mammals, Primates, Vertebrates

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AN 1996:254681 BIOSIS

DN PREV199698810810

TI Mutant p53, decreased thrombospondin-1, and
 angiogenesis may contribute to breast cancer
 progression.

AU Parker, R. J. [Reprint author]; Kyshtoobayeva, A. [Reprint author]; Grant, S.; Fruehauf, J. P. [Reprint author]

CS Oncotech Inc., Irvine, CA 92714, USA

SO Proceedings of the American Association for Cancer Research Annual Meeting, (1996) Vol. 37, No. 0, pp. 83.

Meeting Info.: 87th Annual Meeting of the American Association for Cancer Research. Washington, D.C., USA. April 20-24, 1996.
 ISSN: 0197-016X.

DT Conference; (Meeting)

Conference; Abstract; (Meeting Abstract)

Conference; (Meeting Poster)

LA English

ED Entered STN: 31 May 1996
Last Updated on STN: 31 May 1996

CC General biology - Symposia, transactions and proceedings 00520
Biochemistry studies - Proteins, peptides and amino acids 10064
Cardiovascular system - Blood vessel pathology 14508
Blood - Lymphatic tissue and reticuloendothelial system 15008
Reproductive system - Pathology 16506
Neoplasms - Pathology, clinical aspects and systemic effects 24004
Neoplasms - Carcinogens and carcinogenesis 24007
Development and Embryology - Morphogenesis 25508

IT Major Concepts
Blood and Lymphatics (Transport and Circulation); Cardiovascular
Medicine (Human Medicine, Medical Sciences); Development; Oncology
(Human Medicine, Medical Sciences); Reproductive System (Reproduction)

IT Miscellaneous Descriptors
MEETING ABSTRACT; MEETING POSTER; METASTASIS; ONCOGENESIS; TUMOR GROWTH

ORGN Classifier
Hominidae 86215
Super Taxa
Primates; Mammalia; Vertebrata; Chordata; Animalia
Organism Name
human
Taxa Notes
Animals, Chordates, Humans, Mammals, Primates, Vertebrates

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AN 2005467811 EMBASE

TI Prognostic and predictive molecular markers in DCIS: A review.

AU Nofech-Mozes S.; Spayne J.; Rakovitch E.; Hanna W.

CS W. Hanna, Sunnybrook and Women's College Health Sciences Centre, 2075 Bayview Ave., Toronto, Ont. M4N 3M5, Canada. wedad.hanna@sw.ca

SO Advances in Anatomic Pathology, (2005) Vol. 12, No. 5, pp. 256-264.
Refs: 117
ISSN: 1072-4109

CY United States

DT Journal; General Review

FS 005 General Pathology and Pathological Anatomy
016 Cancer
029 Clinical Biochemistry

LA English

SL English

ED Entered STN: 20051110
Last Updated on STN: 20051110

AB Eighteen percent of all new **breast cancers** detected on screening mammography are ductal carcinoma in situ (DCIS), a preinvasive lesion that is highly curable. However, some women with DCIS will develop life-threatening invasive **breast cancer**. Because the determinants of invasive recurrence are unknown, all women with DCIS require the same treatment (usually with surgery and radiation). Therefore, there is a need to identify biologic markers and create a profile that will provide prognostic information that is more accurate than the currently used van Nuys Index to predict invasive recurrence. In the present review, we examined the many biologic markers studied in **breast cancer**, describe their main biologic role and their expression in DCIS, and review the various studies regarding their ability to serve as prognostic factors in **breast cancer** with an emphasis on predicting invasive recurrence in patients with DCIS. This review covers established markers, namely, ER, PR and HER2/neu, that are used routinely to make treatment decisions as well as investigative biologic factors involved in cell proliferation, cell cycle regulation, extracellular molecules, factors involved in extracellular matrix

degradation, and angiogenesis. However, controversies exist regarding the value of these prognostic factors, their interrelationship, and their advantages over morphologic evaluation. Copyright .COPYRG. 2005 by Lippincott Williams & Wilkins.

CT Medical Descriptors:

- *breast carcinoma: DI, diagnosis
- *breast carcinoma: ET, etiology
- *intraductal carcinoma: DI, diagnosis
- *intraductal carcinoma: ET, etiology
- *carcinoma in situ: DI, diagnosis
- *carcinoma in situ: ET, etiology
- breast disease: DI, diagnosis
- breast disease: ET, etiology
- cancer recurrence
- prediction
- cell cycle
- mitogenesis

angiogenesis

- extracellular matrix
- breast carcinogenesis
- prognosis
- human
- review

priority journal

Drug Descriptors:

- *estrogen receptor: EC, endogenous compound
- *progesterone receptor: EC, endogenous compound
- *epidermal growth factor receptor 2: EC, endogenous compound
- *mitosin: EC, endogenous compound
- *biological marker: EC, endogenous compound
- tumor marker: EC, endogenous compound
- Ki 67 antigen: EC, endogenous compound
- telomerase: EC, endogenous compound
- cyclin D1: EC, endogenous compound
- cyclin A: EC, endogenous compound
- protein p53: EC, endogenous compound
- protein bcl 2: EC, endogenous compound
- protein p21: EC, endogenous compound
- somatomedin binding protein related protein 1: EC, endogenous compound
- cadherin: EC, endogenous compound
- psoriasin: EC, endogenous compound
- urokinase: EC, endogenous compound
- matrix metalloproteinase: EC, endogenous compound
- discoidin: EC, endogenous compound
- discoidin domain receptor: EC, endogenous compound
- CD31 antigen: EC, endogenous compound
- CD34 antigen: EC, endogenous compound
- blood clotting factor 8: EC, endogenous compound
- cyclooxygenase 2: EC, endogenous compound
- thrombospondin 1: EC, endogenous compound
- messenger RNA
- complementary DNA
- unclassified drug

RN (epidermal growth factor receptor 2) 137632-09-8; (protein bcl 2) 219306-68-0; (protein p21) 85306-28-1; (urokinase) 139639-24-0; (discoidin) 81669-85-4, 81669-86-5; (blood clotting factor 8) 9001-27-8; (thrombospondin 1) 343987-56-4

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AN 2005170453 EMBASE

TI Small interfering RNA for experimental cancer therapy.

AU Tong A.W.; Zhang Y.-A.; Nemunaitis J.

CS A.W. Tong, Mary Crowley Medical Research Center, 3500 Gaston Avenue,

Dallas, TX 75246, United States. alext@baylorhealth.edu

SO Current Opinion in Molecular Therapeutics, (2005) Vol. 7, No. 2, pp. 114-124.
 Refs: 98
 ISSN: 1464-8431 CODEN: CUOTFO

CY United Kingdom

DT Journal; General Review

FS 004 Microbiology
 016 Cancer
 022 Human Genetics
 030 Pharmacology
 037 Drug Literature Index
 039 Pharmacy

LA English

SL English

ED Entered STN: 20050428
 Last Updated on STN: 20050428

AB RNA interference describes the recently discovered process of sequence-specific, post-transcriptional gene silencing that is initiated by double-stranded RNA molecules known as small interfering RNAs (siRNAs). siRNAs have an acceptable half-life in vitro, a predictable biodistribution profile similar to that of single-stranded antisense oligonucleotides (ASOs), and have repeatedly been more robust than ASO techniques in terms of consistency of transcript knockdown and threshold concentration. Following validation in mammalian cells by Tuschl and co-workers in 2001, synthetic siRNAs have gained wide acceptance as a laboratory tool for target validation. Currently, there is considerable interest in the therapeutic use of siRNA, particularly in areas of infectious disease and cancer. In vitro and in vivo findings demonstrate the efficacy of siRNA knockdown of gene messages that are pivotal for tumor cell growth, metastasis, angiogenesis and chemoresistance, leading to tumor growth suppression. However, siRNA-based cancer therapy faces similar pharmacokinetic limitations to ASO therapy with respect to the extent that siRNA accesses primary and metastatic target cells. The recently identified 'off-target activity' of siRNAs is also of concern. The concept of carrier-restricted delivery of siRNA by conditionally replicative, oncolytic adenoviruses is discussed. Oncolytic adenoviral delivery offers the potential benefits of restricted and renewable siRNA expression within the tumor microenvironment, an additive antitumor outcome through viral oncolysis and siRNA-mediated oncogene silencing, and a proven clinical platform with respect to infectivity and safety. .COPYRGT. The Thomson Corporation.

CT Medical Descriptors:
 RNA interference
 posttranscriptional gene silencing
 drug half life
 drug distribution
 validation process
 drug efficacy
 tumor growth
 drug targeting
 metastasis
 tumor vascularization
 cancer resistance
 cancer inhibition
 adenovirus vector
 viral gene delivery system
 antineoplastic activity
 oncolytic virus
 oncogene
 drug safety
 virus infectivity
 treatment outcome
 autoimmune hepatitis: DT, drug therapy

breast cancer: DT, drug therapy
 pancreas adenocarcinoma: DT, drug therapy
 drug specificity
 retrovirus vector
 drug potentiation
 drug tolerability
 solid tumor: DT, drug therapy
 dose response
 plasmid vector
 drug design
 viral gene therapy
 glioma: DT, drug therapy
 lentivirus vector
 genetic transduction
 human
 nonhuman
 clinical trial
 review
 Drug Descriptors:
 *small interfering RNA: CT, clinical trial
 *small interfering RNA: CB, drug combination
 *small interfering RNA: CM, drug comparison
 *small interfering RNA: DV, drug development
 *small interfering RNA: IT, drug interaction
 *small interfering RNA: DT, drug therapy
 *small interfering RNA: PR, pharmaceuticals
 *small interfering RNA: PK, pharmacokinetics
 *small interfering RNA: PD, pharmacology
 *small interfering RNA: IP, intraperitoneal drug administration
 *small interfering RNA: TU, intratumoral drug administration
 *small interfering RNA: IV, intravenous drug administration
 *small interfering RNA: VI, intravitreal drug administration
 antisense oligonucleotide: CT, clinical trial
 antisense oligonucleotide: CM, drug comparison
 antisense oligonucleotide: DO, drug dose
 antisense oligonucleotide: DT, drug therapy
 antisense oligonucleotide: PK, pharmacokinetics
 antisense oligonucleotide: PD, pharmacology
 ribozyme: CT, clinical trial
 ribozyme: CM, drug comparison
 ribozyme: DT, drug therapy
 ribozyme: PD, pharmacology
 ribozyme: SC, subcutaneous drug administration
 liposome: PR, pharmaceuticals
 double stranded RNA: DT, drug therapy
 double stranded RNA: PR, pharmaceuticals
 double stranded RNA: PD, pharmacology
 double stranded RNA: IP, intraperitoneal drug administration
 double stranded RNA: TU, intratumoral drug administration
 short hairpin RNA: DT, drug therapy
 short hairpin RNA: PR, pharmaceuticals
 short hairpin RNA: PD, pharmacology
 short hairpin RNA: TU, intratumoral drug administration
 short hairpin RNA: IV, intravenous drug administration
 gemcitabine: CB, drug combination
 gemcitabine: IT, drug interaction
 gemcitabine: DT, drug therapy
 gemcitabine: PD, pharmacology
 thrombospondin 1: CB, drug combination
 thrombospondin 1: IT, drug interaction
 thrombospondin 1: DT, drug therapy
 thrombospondin 1: PD, pharmacology
 sirna 027: CT, clinical trial
 sirna 027: DT, drug therapy

sirna 027: VI, intravitreal drug administration
 angiozyme: CT, clinical trial
 angiozyme: CM, drug comparison
 angiozyme: DT, drug therapy
 angiozyme: PD, pharmacology
 angiozyme: SC, subcutaneous drug administration
 immunoliposome: PR, pharmaceuticals
 protein p53: DT, drug therapy
 protein p53: PR, pharmaceuticals
 protein p53: PD, pharmacology
 protein p53: TU, intratumoral drug administration
 advexin: DT, drug therapy
 advexin: PR, pharmaceuticals
 advexin: PD, pharmacology
 advexin: TU, intratumoral drug administration
 ONYX 015: DT, drug therapy
 ONYX 015: PR, pharmaceuticals
 ONYX 015: PD, pharmacology
 ONYX 015: IA, intraarterial drug administration
 ONYX 015: TU, intratumoral drug administration
 antineoplastic agent: CT, clinical trial
 antineoplastic agent: CB, drug combination
 antineoplastic agent: CM, drug comparison
 antineoplastic agent: DV, drug development
 antineoplastic agent: DO, drug dose
 antineoplastic agent: IT, drug interaction
 antineoplastic agent: DT, drug therapy
 antineoplastic agent: PR, pharmaceuticals
 antineoplastic agent: PK, pharmacokinetics
 antineoplastic agent: PD, pharmacology
 antineoplastic agent: IA, intraarterial drug administration
 antineoplastic agent: IP, intraperitoneal drug administration

CT Drug Descriptors:

antineoplastic agent: TU, intratumoral drug administration
 antineoplastic agent: IV, intravenous drug administration
 antineoplastic agent: VI, intravitreal drug administration
 antineoplastic agent: SC, subcutaneous drug administration
 onyx 411: CB, drug combination
 onyx 411: IT, drug interaction
 onyx 411: DT, drug therapy
 onyx 411: PD, pharmacology
 onyx 411: IV, intravenous drug administration
 onyx 443: DT, drug therapy
 onyx 443: PD, pharmacology
 onyx 443: IV, intravenous drug administration
 ONYX 321: PD, pharmacology
 unclassified drug

RN (gemcitabine) 103882-84-4; (thrombospondin 1)
343987-56-4

CN (1) Sirna 027; (2) Ingn 201

CO (1) Sirna therapeutics; (2) Introgen

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AN 2004350415 EMBASE

TI Gene-based therapy in prostate cancer.

AU Foley R.; Lawler M.; Hollywood D.

CS Prof. D. Hollywood, Department of Haematology/Oncology, Institute of Molecular Medicine, St. James' Hospital/Trinity College, Dublin 8, Ireland. dhlywood@tcd.ie

SO Lancet Oncology, (1 Aug 2004) Vol. 5, No. 8, pp. 469-479.

Refs: 75

ISSN: 1470-2045 CODEN: LOANBN

PUI S 1470-2045(04)01525-6

CY United States
 DT Journal; General Review
 FS 016 Cancer
 022 Human Genetics
 028 Urology and Nephrology
 037 Drug Literature Index
 038 Adverse Reactions Titles
 039 Pharmacy
 LA English
 SL English
 ED Entered STN: 20040902
 Last Updated on STN: 20040902
 AB Prostate cancer is one of the commonest causes of illness and death from cancer. Radical prostatectomy, radiotherapy, and hormonal therapy are the main conventional treatments. However, gene therapy is emerging as a promising adjuvant to conventional strategies, and several clinical trials are in progress. Here, we outline several approaches to gene therapy for prostate cancer that have been investigated. Methods of gene delivery are described, particularly those that have commonly been used in research on prostate cancer. We discuss efforts to achieve tissue-specific gene delivery, focusing on the use of tissue-specific gene promoters. Finally, the present use of gene therapy for prostate cancer is evaluated. The ability to deliver gene-therapy vectors directly to prostate tissue, and to regulate gene expression in a tissue-specific manner, offers promise for the use of gene therapy in prostate cancer.
 CT Medical Descriptors:
 *gene therapy
 *prostate cancer: DT, drug therapy
 *prostate cancer: PC, prevention
 *prostate cancer: RT, radiotherapy
 *prostate cancer: SU, surgery
 morbidity
 cause of death
 cancer mortality
 prostatectomy
 cancer radiotherapy
 cancer hormone therapy
 cancer adjuvant therapy
 viral gene delivery system
 nonviral gene delivery system
 cancer research
 tissue specificity
 gene expression regulation
 drug mechanism
 suicide gene therapy
 promoter region
 cancer immunotherapy
 thrombocytopenia: SI, side effect
 lymphocytopenia: SI, side effect
 human
 nonhuman
 clinical trial
 review
 priority journal
 Drug Descriptors:
 *antineoplastic agent: AE, adverse drug reaction
 *antineoplastic agent: CT, clinical trial
 *antineoplastic agent: CB, drug combination
 *antineoplastic agent: DT, drug therapy
 *antineoplastic agent: PR, pharmaceuticals
 *antineoplastic agent: PD, pharmacology
 *antineoplastic agent: DL, intradermal drug administration

*antineoplastic agent: IM, intramuscular drug administration
 *antineoplastic agent: IV, intravenous drug administration
 *antineoplastic agent: SC, subcutaneous drug administration
 antisense oligonucleotide: CT, clinical trial
 antisense oligonucleotide: DT, drug therapy
 antisense oligonucleotide: TO, drug toxicity
 antisense oligonucleotide: PR, pharmaceuticals
 antisense oligonucleotide: PD, pharmacology
 antisense oligonucleotide: IV, intravenous drug administration
 oligonucleotide: PD, pharmacology
 small interfering RNA: PD, pharmacology
 double stranded DNA: PD, pharmacology
 thymidine kinase: AE, adverse drug reaction
 thymidine kinase: CT, clinical trial
 thymidine kinase: CB, drug combination
 thymidine kinase: DT, drug therapy
 thymidine kinase: PR, pharmaceuticals
 thymidine kinase: PD, pharmacology
 ganciclovir: CT, clinical trial
 ganciclovir: CB, drug combination
 ganciclovir: DT, drug therapy
 ganciclovir: PR, pharmaceuticals
 ganciclovir: PD, pharmacology
 tumor suppressor protein: AE, adverse drug reaction
 tumor suppressor protein: CT, clinical trial
 tumor suppressor protein: DT, drug therapy
 tumor suppressor protein: PR, pharmaceuticals
 tumor suppressor protein: PD, pharmacology
 tumor suppressor protein: TU, intratumoral drug administration
 protein p53: DT, drug therapy
 protein p53: PR, pharmaceuticals
 protein p53: PD, pharmacology
 protein Bax: PR, pharmaceuticals
 protein Bax: PD, pharmacology
 angiogenesis inhibitor: DT, drug therapy
 angiogenesis inhibitor: PR, pharmaceuticals
 angiogenesis inhibitor: PD, pharmacology
 thrombospondin 1: DT, drug therapy
 thrombospondin 1: PR, pharmaceuticals
 thrombospondin 1: PD, pharmacology
 cytokine: DT, drug therapy
 cytokine: PR, pharmaceuticals
 cytokine: PD, pharmacology
 interleukin 2: AE, adverse drug reaction
 interleukin 2: CT, clinical trial
 interleukin 2: DT, drug therapy
 interleukin 2: PR, pharmaceuticals
 interleukin 2: PD, pharmacology
 interleukin 2: TU, intratumoral drug administration
 tumor antigen: DT, drug therapy
 tumor antigen: PR, pharmaceuticals
 tumor antigen: PD, pharmacology
 tumor antigen: DL, intradermal drug administration
 tumor antigen: IM, intramuscular drug administration
 tumor antigen: SC, subcutaneous drug administration
 prostate specific antigen: AE, adverse drug reaction
 prostate specific antigen: CT, clinical trial
 prostate specific antigen: DT, drug therapy
 prostate specific antigen: PR, pharmaceuticals
 prostate specific antigen: PD, pharmacology
 prostate specific antigen: DL, intradermal drug administration
 prostate specific antigen: IM, intramuscular drug administration
 prostate specific antigen: SC, subcutaneous drug administration
 cytosine deaminase: AE, adverse drug reaction

cytosine deaminase: CT, clinical trial
 cytosine deaminase: CB, drug combination
 cytosine deaminase: DT, drug therapy
 cytosine deaminase: PR, pharmaceuticals
 cytosine deaminase: PD, pharmacology
 flucytosine: CB, drug combination
 flucytosine: PR, pharmaceuticals
 flucytosine: PD, pharmacology
 valaciclovir: CT, clinical trial
 valaciclovir: CB, drug combination
 valaciclovir: DT, drug therapy
 valaciclovir: PR, pharmaceuticals
 valaciclovir: PD, pharmacology
 caspase 9: PR, pharmaceuticals
 caspase 9: PD, pharmacology
 diphtheria toxin: DT, drug therapy
 diphtheria toxin: EC, endogenous compound
 CT Drug Descriptors:
 diphtheria toxin: PR, pharmaceuticals
 diphtheria toxin: PD, pharmacology
 granulocyte macrophage colony stimulating factor: CT, clinical trial
 granulocyte macrophage colony stimulating factor: DT, drug therapy
 granulocyte macrophage colony stimulating factor: PR, pharmaceuticals
 granulocyte macrophage colony stimulating factor: PD, pharmacology
 granulocyte macrophage colony stimulating factor: DL, intradermal drug administration
 transforming growth factor beta receptor: DT, drug therapy
 transforming growth factor beta receptor: PD, pharmacology
 mutant protein: DT, drug therapy
 mutant protein: PD, pharmacology
 docetaxel: DT, drug therapy
 docetaxel: TO, drug toxicity
 probasin: DT, drug therapy
 protein bcl 2: DT, drug therapy
 kallikrein: DT, drug therapy
 gamma glutamyl hydrolase: DT, drug therapy
 unindexed drug
 RN (thymidine kinase) 9002-06-6, 9086-73-1; (ganciclovir) 82410-32-0; (**thrombospondin** 1) 343987-56-4; (interleukin 2) 85898-30-2; (cytosine deaminase) 9025-05-2; (flucytosine) 2022-85-7; (valaciclovir) 124832-26-4; (caspase 9) 180189-96-2; (docetaxel) 114977-28-5; (protein bcl 2) 219306-68-0; (kallikrein) 8006-48-2, 9001-01-8; (gamma glutamyl hydrolase) 55326-32-4, 9074-87-7
 L14 ANSWER 23 OF 26 EMBASE COPYRIGHT (c) 2005 Elsevier B.V. All rights reserved on STN
 AN 2001374392 EMBASE
 TI Review: Molecular pathology of cyclooxygenase-2 in cancer-induced **angiogenesis**.
 AU Fosslie E.
 CS Dr. E. Fosslie, Department of Pathology (M/C 847), College of Medicine, University of Illinois at Chicago, 1819 West Polk Street, Chicago, IL 60612, United States. efosslie@uic.edu
 SO Annals of Clinical and Laboratory Science, (2001) Vol. 31, No. 4, pp. 325-348.
 Refs: 169
 ISSN: 0091-7370 CODEN: ACLSCP
 CY United States
 DT Journal; General Review
 FS 005 General Pathology and Pathological Anatomy
 016 Cancer
 029 Clinical Biochemistry
 030 Pharmacology
 037 Drug Literature Index

LA English
 SL English
 ED Entered STN: 20011108
 Last Updated on STN: 20011108
 AB Cancer-induced **angiogenesis** is the result of increased expression of angiogenic factors, or decreased expression of anti-angiogenic factors, or a combination of both events. For instance, in colon cancer, the malignant cells, the stromal fibroblasts, and the endothelial cells all exhibit strong staining for cyclooxygenase-2 (COX-2), the rate-controlling enzyme in prostaglandin (PG) synthesis. In various cancer tissues, vascular endothelial growth factor (VEGF) and transforming growth factor β (TGF- β) co-localize with COX-2. Strong COX-2 and VEGF expression is highly correlated with increased tumor microvascular density (MCD); new vessels proliferate in areas of the tumor that express COX-2. Moreover, high MVD is a predictor of poor prognosis in breast and cervical cancers. COX-2 and VEGF expression are elevated in breast and prostate cancer tissues and their cell-lines. In vitro, PGE2 induces VEGF. Supernatants of cultured cells from breast, prostate, and squamous cell cancers contain angiogenic proteins such as COX-2 and VEGF that induce in vitro **angiogenesis**. A selective COX-2 inhibitor, NS-398, restores tumor cell apoptosis, reduces microvascular density, and reduces tumor growth of PC-3 prostate carcinoma cells xenografted into nude mice. The COX-2 produced by a malignant tumor and COX-2 produced by the surrounding host tissue both contribute to new vessel formation, which explains how selective COX-2 inhibition reduces tumor growth where the tumor COX-2 gene has been silenced by methylation.

CT Medical Descriptors:
 ***angiogenesis**
 *tumor vascularization
 molecular biology
 microvascularization
 stroma cell
 fibroblast
 endothelium cell
 prostaglandin synthesis
 colon cancer: ET, etiology
 breast cancer: ET, etiology
 prostate cancer: ET, etiology
 uterine cervix cancer: ET, etiology
 squamous cell carcinoma: ET, etiology
 in vitro study
 apoptosis
 cancer inhibition
 carcinogenesis
 antineoplastic activity
 human
 nonhuman
 review
 priority journal
 Drug Descriptors:
 *cyclooxygenase 2: EC, endogenous compound
 vasculotropin: EC, endogenous compound
 transforming growth factor beta: EC, endogenous compound
 n (2 cyclohexyloxy 4 nitrophenyl)methanesulfonamide: PD, pharmacology
 celecoxib: PD, pharmacology
 rofecoxib: PD, pharmacology
 nonsteroid antiinflammatory agent: PD, pharmacology
 protein p53: EC, endogenous compound
 prostaglandin E2: EC, endogenous compound
 nitric oxide synthase: EC, endogenous compound
 endoglin: EC, endogenous compound
 4 (4 cyclohexyl 2 methyl 5 oxazolyl) 2 fluorobenzenesulfonamide: PD, pharmacology

haptoglobin: EC, endogenous compound

thrombospondin 1: EC, endogenous compound

angiotatin: EC, endogenous compound

metalloproteinase inhibitor: EC, endogenous compound

CD31 antigen: EC, endogenous compound

RN (vasculotropin) 127464-60-2; (n (2 cyclohexyloxy 4
nitrophenyl)methanesulfonamide) 123653-11-2; (celecoxib) 169590-42-5;
(rofecoxib) 162011-90-7, 186912-82-3; (prostaglandin E2) 363-24-6; (nitric
oxide synthase) 125978-95-2; (4 (4 cyclohexyl 2 methyl 5 oxazolyl) 2
fluorobenzenesulfonamide) 180200-68-4; (haptoglobin) 9087-69-8;
(angiotatin) 172642-30-7, 86090-08-6

CN Ns 398; Jte 522

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AN 2001087714 EMBASE

TI Expression of **thrombospondin-1** in pancreatic
carcinoma: Correlation with microvessel density.

AU Kasper H.U.; Ebert M.; Malfertheiner P.; Roessner A.; Kirkpatrick C.J.;
Wolf H.K.

CS H.U. Kasper, Department of Pathology, Otto-von-Guericke University,
Leipziger Strasse 44, 39112 Magdeburg, Germany. hukasper@hotmail.com

SO Virchows Archiv, (2001) Vol. 438, No. 2, pp. 116-120.

Refs: 38

ISSN: 0945-6317 CODEN: VARCEM

CY Germany

DT Journal; Article

FS 016 Cancer

048 Gastroenterology

LA English

SL English

ED Entered STN: 20010406

Last Updated on STN: 20010406

AB **Thrombospondin-1** (TSP-1) is a multifunctional platelet
and extracellular matrix protein that is involved in **angiogenesis**
. Under certain pathological conditions, e.g., malignant tumors, high
concentrations of TSP-1 work as an angiogenic agonist. Here we examined
98 pancreatic carcinomas with respect to TSP-1 immunoreactivity and its
correlation to intratumoral microvessel density (MVD), a representation of
the overall degree of **angiogenesis** in carcinomas. Northern blot
analysis for TSP-1 mRNA was performed in seven additional cases.
Eighty-seven tumors showed strong TSP-1 immunoreactivity, nine carcinomas
were only weakly positive, and two lesions were negative for TSP-1. TSP-1
immunoreactivity was detected in the extracellular matrix, mostly at the
invasion front of the tumor. Using Northern blot analysis, we observed
high levels of TSP-1 mRNA in three out of seven pancreatic carcinomas.
The mean MVD in pancreatic carcinoma was 38.8 vessels per mm². Tumors
with a high expression of TSP-1 showed a higher MVD and the correlation
between TSP-1 immunoreactivity and microvessel density was highly
significant (P=0.003). As a modulator of **angiogenesis**, TSP-1 is
strongly expressed in most pancreatic adenocarcinomas and is likely to
contribute to the extensive neovascularization and spread of this highly
aggressive tumor.

CT Medical Descriptors:

*pancreas cancer: DI, diagnosis

***angiogenesis**

*gene expression

Northern blotting

immunoreactivity

extracellular matrix

neovascularization (pathology)

thrombocyte

prognosis

endometrium cancer: DI, diagnosis

breast cancer: DI, diagnosis
ovary cancer: DI, diagnosis
colon cancer: DI, diagnosis
lung adenocarcinoma: DI, diagnosis
tumor suppressor gene
human
major clinical study
human tissue
human cell
article
priority journal
Drug Descriptors:
 *thrombospondin 1
messenger RNA
disulfide
 protein p53
protein p16

RN (disulfide) 16734-12-6

L14 ANSWER 25 OF 26 EMBASE COPYRIGHT (c) 2005 Elsevier B.V. All rights reserved on STN

AN 1998333116 EMBASE

TI Gene therapy with P53 and a fragment of thrombospondin I inhibits human breast cancer in vivo.

AU Xu M.; Kumar D.; Stass S.A.; Mixson A.J.

CS A.J. Mixson, Department of Pathology, University of Maryland, Building MSTF, 10 S. Pine Street, Baltimore, MD 21201, United States

SO Molecular Genetics and Metabolism, (1998) Vol. 63, No. 2, pp. 103-109.
Refs: 24

ISSN: 1096-7192 CODEN: MGMEFF

CY United States

DT Journal; Article

FS 016 Cancer

022 Human Genetics

030 Pharmacology

037 Drug Literature Index

LA English

SL English

ED Entered STN: 19981028

Last Updated on STN: 19981028

AB We recently reported that a p53 encoding plasmid (BAP-p53) complexed to liposomes administered intravenously markedly attenuates the growth of a malignant human breast tumor. We now have found that systemically delivered liposomes complexed to a plasmid expressing an established antiangiogenic peptide of thrombospondin I (BAP-TSPf) decreased the growth of MDA-MB-435 tumors compared to controls in nude mice. Compared to BAP-p53, the BAP-TSPf group had a similar antitumor efficacy. More importantly, liposomes complexed with BAP-TSPf and BAP-p53 synergistically decreased the growth of MDA-MB-435 tumors when compared to either BAP-p53 or BAP-TSPf alone. Furthermore, we also determined that the combination therapy of p53 and TSPf inhibited endothelial cells in vitro more than either p53 or TSPf alone. There was also a significant decrease of the blood vessel density in the combination p53 and TSPf treatment group compared to the control groups. These results suggest that liposomes complexed to a tumor suppressor and antiangiogenic genes may be effective in treating metastatic tumors.

CT Medical Descriptors:

*gene therapy

*breast cancer: TH, therapy

plasmid

antineoplastic activity

tumor growth

angiogenesis

endothelium cell
 tumor suppressor gene
 metastasis
 nonhuman
 mouse
 animal model
 controlled study
 animal tissue
 article
 priority journal
 Drug Descriptors:
 *liposome: PD, pharmacology
 *protein p53: PD, pharmacology
 *thrombospondin 1: PD, pharmacology

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DN 1997352894

TI Evidence of a dominant transcriptional pathway which regulates an undifferentiated and complete metastatic phenotype.

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013 Dermatology and Venereology

016 Cancer

022 Human Genetics

LA English

SL English

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AB The highly metastatic amelanotic C8161 human melanoma line was found to exhibit complete dominance of its undifferentiated and metastatic phenotype in multiple somatic cell hybridization studies designed to bypass the presence of potential tumor suppressor genes. In a three armed approach involving somatic cell fusions of C8161 with recipient lines of greater differentiation, different lineage, and different tumorigenicity status, the metastatic and undifferentiated phenotype of C8161 was promiscuously dominant. In somatic cell hybrids produced between the C8161 and a group of non-metastatic human melanoma lines which exhibited melanocyte differentiation markers including S100, HMB-45, NKI/C3, aC3, and melanin, the fusions were uniformly metastatic and undifferentiated. In somatic cell hybrids of C8161 and MCF-7 the fusions exhibited an estrogen independent and unresponsive, estrogen receptor (ER) negative, and highly metastatic phenotype. In fusions between C8161 and HMS-1, an immortalized 'benign' human myoepithelial line which produced an abundant extracellular matrix (ECM) and high levels of protease and angiogenic inhibitors including maspin, tissue inhibitor of metalloproteinase-1 (TIMP-1), α 1-antitrypsin (α 1-AT), protease nexin II (PN-II), thrombospondin-1 and soluble basic fibroblast growth factor (bFGF) receptors, the hybrids showed complete absence of matrix, absent maspin expression, markedly decreased protease inhibitor and angiogenic inhibitor production, high levels of proteases and angiogenic factors, and a highly metastatic phenotype. In our somatic cell fusions, the human-human hybrids represented true and complete fusions and not hybrid clones selected for by loss of dominant-acting growth suppressor genes. This finding was supported by detailed

comparative genomic hybridization (CGH) studies, Q-banding karyotype analysis, and autofusions of representative clones. The purposeful creation of inherently unstable human-murine fusions between C8161 and B16-F1 where loss of putative suppressor loci would be expected, resulted in fusions exhibiting decreased growth and non-metastatic behavior with progressive chromosomal loss. Neither p53, nm23, DNA methyltransferase, activated ras, fibroblast growth factor-4 (FGF-4), or epidermal growth factor receptor (EGFR) mediated the acquisition of the metastatic or undifferentiated phenotype within the C8161-human fusions. These studies are the first studies ever to successfully transfer the complete metastatic phenotype by somatic cell fusion and support the presence of a new high level regulatory pathway(s) involving dominant trans-acting factors which act pleiotropically to regulate an undifferentiated and highly metastatic phenotype.

CT Medical Descriptors:

*metastasis
*transcription regulation

animal cell

article

cell clone

cell differentiation

chromosome loss

controlled study

extracellular matrix

gene locus

genetic transcription

human

human cell

hybrid cell

karyotyping

melanocyte

 melanoma

mouse

nonhuman

phenotype

priority journal

somatic cell

tumor suppressor gene

Drug Descriptors:

alpha 1 antitrypsin

 angiogenesis inhibitor

basic fibroblast growth factor

dna methyltransferase: EC, endogenous compound

epidermal growth factor receptor: EC, endogenous compound

estrogen

estrogen receptor

fibroblast growth factor 4: EC, endogenous compound

fibroblast growth factor receptor

protease nexin

 protein p53: EC, endogenous compound

proteinase inhibitor

ras protein: EC, endogenous compound

thrombospondin

tissue inhibitor of metalloproteinase

trans acting factor: EC, endogenous compound

RN (alpha 1 antitrypsin) 9041-92-3; (basic fibroblast growth factor)

106096-93-9; (dna methyltransferase) 9037-42-7; (proteinase inhibitor)

37205-61-1; (tissue inhibitor of metalloproteinase) 97837-28-0